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Case Report

Complete atrioventricular block associated with not apical but midventricular ballooning

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ABSTRACT

An 86-year-old woman was admitted to hospital with a head injury secondary to an episode of syncope associated with incontinence. Electrocardiography showed complete atrioventricular block, giant negative T waves in the precordial leads, and QT interval prolongation. Emergency coronary angiography showed no significant coronary stenosis, while left ventriculography demonstrated midventricular ballooning. Despite temporary transvenous pacing, her complete atrioventricular block persisted, but worsening of heart failure did not occur. Although left ventricular wall motion improved, complete atrioventricular block remained, so a pacemaker was implanted on day 18 after admission. There have been no previous reports of complete atrioventricular block associated with midventricular ballooning. This case demonstrates that complete atrioventricular block may persist after improvement of left ventricular wall motion in patients with midventricular ballooning and implantation of a pacemaker may be needed.
<**Learning objective:** Some cases describe takotsubo-like cardiomyopathy with not apical but midventricular ballooning. This is the first case of midventricular ballooning and persistence of complete atrioventricular block after improvement of left ventricular wall motion that required implantation of a pacemaker.>

Introduction

Transient left ventricular dysfunction associated with chest symptoms and electrocardiographic (ECG) changes mimicking those of acute myocardial infarction is known as takotsubo cardiomyopathy or transient left ventricular apical ballooning syndrome [1–3]. Although the pathogenesis of this type of cardiomyopathy remains unclear, physical or emotional stress is common in these patients and elevation of plasma catecholamines suggests that sympathetic stimulation plays a central role. When treatment is prompt, the long-term prognosis of takotsubo cardiomyopathy is generally good with full recovery of left ventricular contraction. Patients generally show T-wave inversion and QT interval prolongation during the early phase, but these ECG changes typically normalize within several weeks along with improvement in left ventricular wall motion [1–3]. The arrhythmias associated with takotsubo cardiomyopathy include atrioventricular (AV) block, sinus bradycardia, paroxysmal atrial fibrillation, ventricular tachycardia, and ventricular fibrillation [2–9]. Here, we describe the first case of takotsubo-like cardiomyopathy with midventricular ballooning and persistence of complete AV block after improvement in left ventricular wall motion that required implantation of a pacemaker.

Case report

An 86-year-old woman without any significant medical history became unsteady, followed by loss of consciousness and a head injury when she fell. She was then transported to our emergency room.

Physical examination was unremarkable and her consciousness was clear at the time of reaching hospital. The serum level of creatine kinase was 41 U/ml (45–163 IU/l) and troponin I was 0.07 ng/ml (0.00–0.09 ng/ml). The initial ECG revealed complete AV block with escape rhythm of incomplete right bundle branch block (IRBBB), giant negative T waves in leads V1–V4, and QT prolongation. Escape rhythm rate was 46 bpm and QRS complexes were 110 ms (Fig. 1).

Several hours later, the ECG showed advanced AV block and IRBBB, as well as inversion of T waves in leads V1–V4 (Fig. 2).

No previous ECG results were available.

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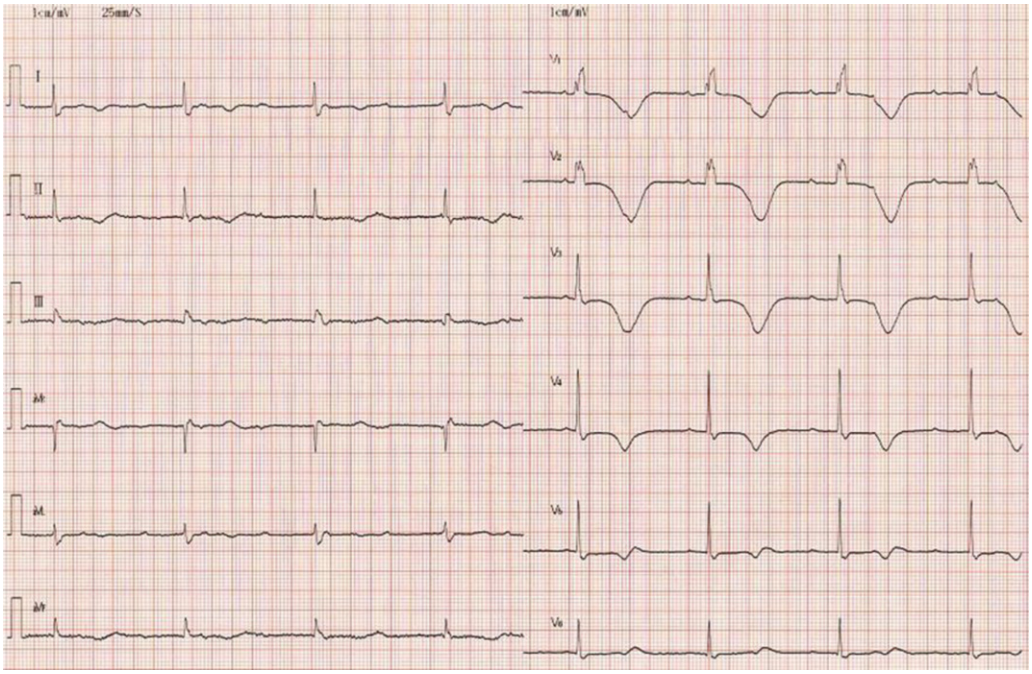

Fig. 1. Initial electrocardiogram showing complete AV block with incomplete right bundle branch block, giant negative T waves in leads V1–V4, and a prolonged QT interval. Escape rhythm rate was 46 bpm and QRS complexes were 110 ms.

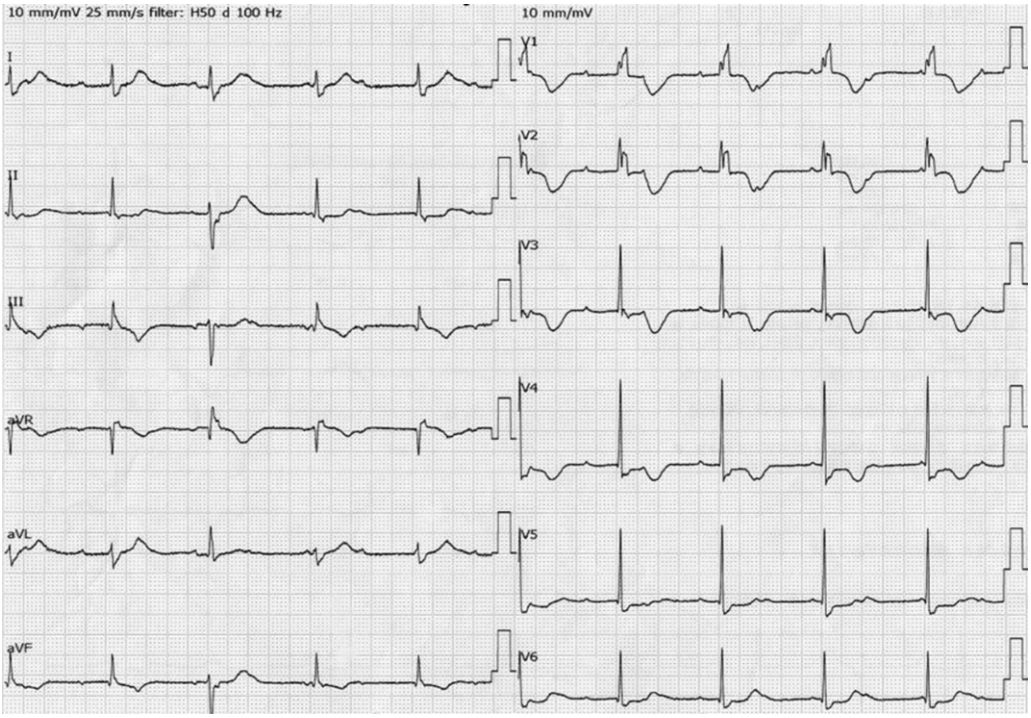

Fig. 2. Repeat electrocardiogram. Several hours after the first recording, there were advanced atrioventricular block, incomplete right bundle branch block, and inversion of T waves in leads V1–V4.

Echocardiography after admission revealed midventricular ballooning with a left ventricular ejection fraction of 50% and mild mitral regurgitation. Temporary right ventricular pacing was initiated. Urgent coronary angiography was performed, revealing normal coronary arteries (Fig. 3), while left ventriculography confirmed midventricular ballooning and apical akinesis (Fig. 4). After 10 days in hospital, her systolic function gradually improved and repeat echocardiography showed normalization of regional contractility with an ejection fraction of 61%. However, her complete AV block changed to advanced AV block, so a permanent pacemaker was implanted after 18 days. Nuclear imaging was performed after two weeks. There were no definite abnormalities on thallium scintigraphy. However, I-123 betamethyl-p-iodophenylpentadecanoic acid (I-123 BMIPP) uptake was reduced at the mid-portion of the left ventricle where akinesis was shown by left ventriculography (Fig. 5). The patient was discharged in a stable condition. One year later, her pacemaker is operating well (the frequency of atrial pacing is 5%, the frequency of ventricular pacing is 100%). Although the ECG has not returned to normal sinus rhythm, uptake has improved on scintigraphy (Fig. 6).

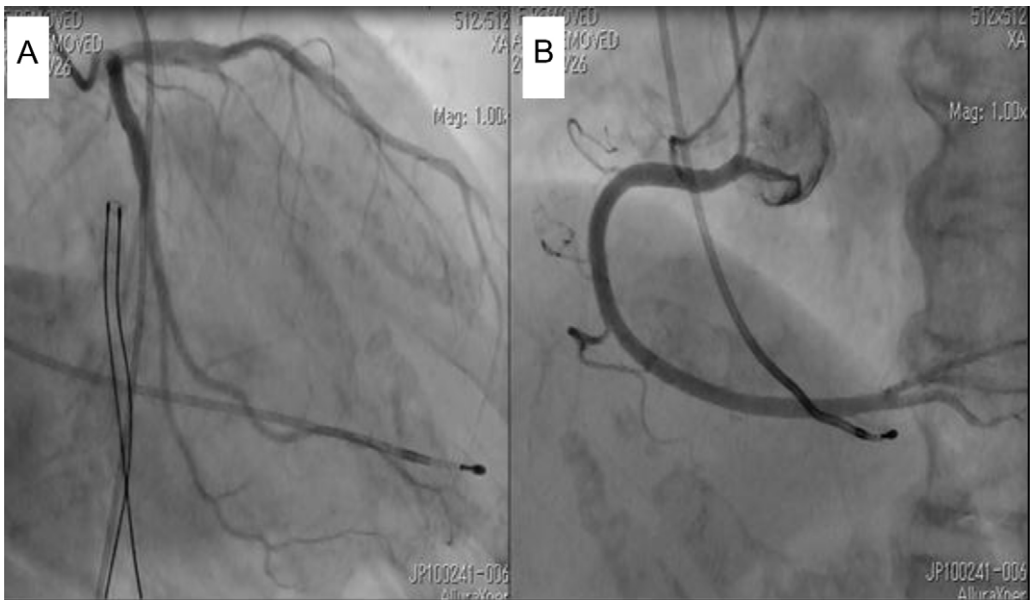

Fig. 3. Coronary angiography. Coronary angiography was performed urgently, but showed normal coronary arteries. (A) Left coronary artery and (B) right coronary artery.

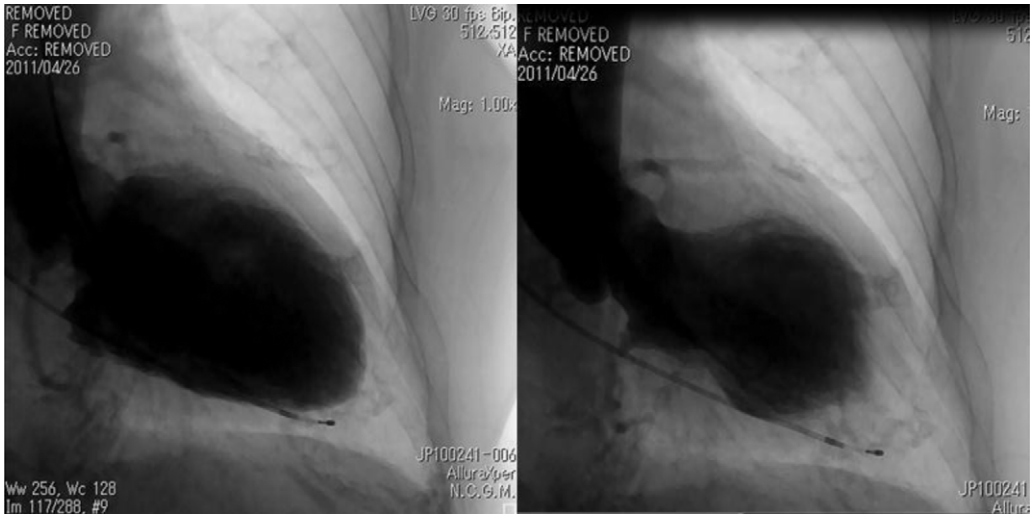

Fig. 4. Left ventriculography. Left ventriculography showed midventricular akinesis and apical normokinesis.

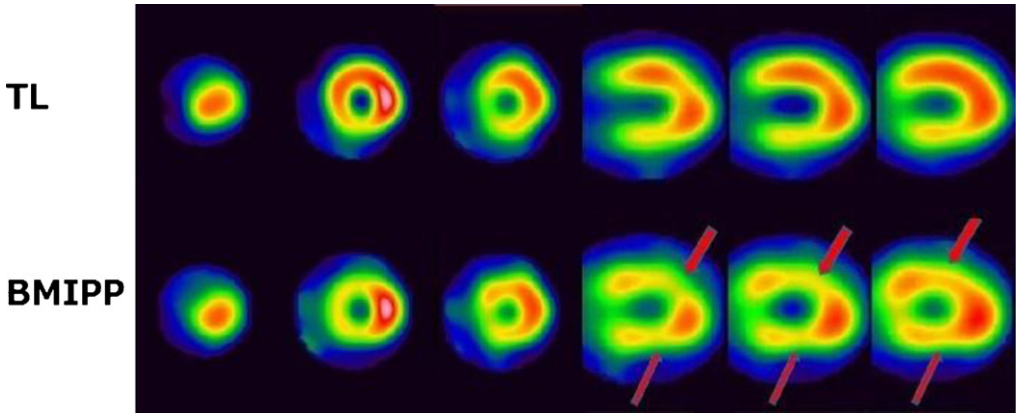

Fig. 5. Nuclear imaging. Resting scintigrams obtained two weeks after hospitalization are shown. There is no abnormal thallium (TL) uptake (upper panel). However, I-123 betamethyl-p-iodophenyl-pentadecanoic acid (BMIPP) uptake is reduced at a region that corresponds to the midventricular akinesis on left ventriculography (lower panel: red arrows). On the other hand, the uptake at apical is normal. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of the article.)

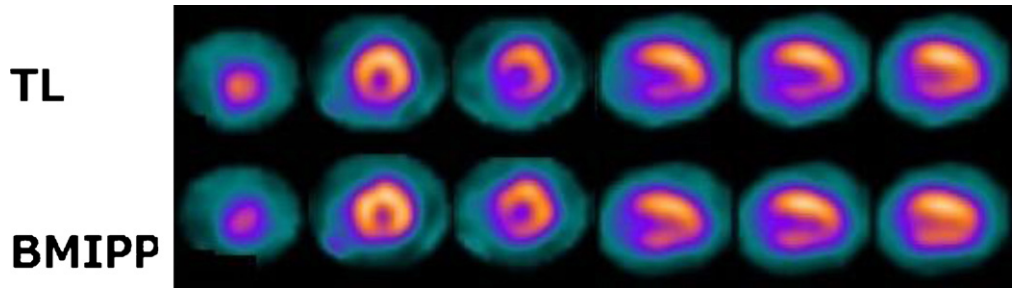

Fig. 6. Nuclear imaging after one year. Resting scintigrams obtained one year after hospitalization are shown. There is no abnormal thallium (TL) uptake (upper panel) and I-123 betamethyl-p-iodophenyl-pentadecanoic acid (BMIPP) uptake (lower panel).

Discussion

Takotsubo cardiomyopathy, or transient left ventricular apical ballooning syndrome, features a unique reversible left ventricular wall motion abnormality, which is characterized as balloon-like left ventricular apex together with hypercontraction of the basal segment. In addition, the ECG shows changes of acute myocardial infarction without any significant stenosis on coronary angiography. This entity was first reported as "takotsubo-like left ventricular dysfunction" by Satoh in 1990, and several other reports followed in Japan [1]. Apical ballooning is often associated with physical or emotional stress, chest pain, and limited release of cardiac injury markers [2].

This syndrome was first described in Japanese patients and about 200 cases have been reported, mainly in women [2–6].

The incidence is unknown because no incidence survey of a regional population has been performed. The diagnostic criteria were first reported by Abe [7]. Then the Mayo criteria were subsequently developed, which include all of the following 4 features: (1) Transient akinesis or dyskinesis of the left ventricular apical and midventricular regions, with abnormal regional wall motion extending beyond a single epicardial vascular territory. (2) No obstructive coronary artery disease or angiographic evidence of acute plaque rupture. (3) New ECG abnormalities (either ST-segment elevation or T-wave inversion). (4) Absence of recent significant head trauma, intracranial bleeding, pheochromocytoma, obstructive epicardial coronary artery disease, myocarditis, and hypertrophic cardiomyopathy [2].

Hurst et al. [8] reported a new variant of this syndrome in four female patients who presented with symptoms suggestive of acute coronary syndrome. Left heart catheterization revealed normal epicardial arteries, while ventriculography showed midventricular ballooning and dilatation along with a hypercontractile apex and base. This variant has a similar clinical presentation and outcome to transient left ventricular apical ballooning. However, it has unique features on echocardiography and left ventriculography that distinguish it, because the apex and base are hypercontractile whereas the midventricular segments show akinesis and ballooning. As with apical ballooning syndrome, the distribution of regional wall motion abnormalities does not fit the epicardial coronary artery territories and ventricular dysfunction is transient. Although the pathogenesis of midventricular ballooning remains unclear, physical or emotional stress is common along with elevation of plasma catecholamines, so sympathetic stimulation may play a major role [1–3].

Our patient had transient heart block combined with midventricular ballooning, which has not been reported before. There were no critical coronary artery lesions, so this syndrome seems unlikely to be associated with ischemia unless there is some microvascular abnormality. Various arrhythmias are associated with left ventricular apical ballooning syndrome and have the potential to be fatal in a proarrhythmic state [8–15]. AV block occurs in 5% of patients with left ventricular apical ballooning syndrome, and there have been several reports of advanced AV block [10,11]. In one patient, 2:1 intra-His block was associated with delayed recovery of left ventricular wall motion and a permanent pacemaker was implanted, but AV conduction became completely normal after 2 years. Another patient had complete AV block lasting for 2 days and received temporary pacing [11]. The specific sites of damage to AV conduction in left ventricular apical ballooning syndrome and midventricular ballooning syndrome are not known. Because the involved segments are located at the apical region and are distant from the atrium and bundle of His, atrio-His block and intra-His block might be rare in left ventricular apical ballooning syndrome. Apical ballooning syndrome tends to be more frequent in elderly women whose AV conduction pathway may be affected by damage related to aging [3,16,17]. It is also possible that stress induced by complete AV block might lead to the onset of apical ballooning syndrome, since there have been several reports of this syndrome occurring a few days after pacemaker implantation for complete AV block [18]. In such cases, it is difficult to judge whether the primary abnormality is apical ballooning itself or complete AV block.

This case suggests that various arrhythmias are associated with midventricular ballooning syndrome. Furthermore, there is some possibility of AV conduction recovery in midventricular ballooning like left ventricular apical ballooning syndrome.

The optimum management of complete AV block associated with left ventricular apical ballooning syndrome remains unclear since there is no information about how long AV block lasts and whether it is transient or persistent.

Despite improvement in left ventricular wall motion, AV conduction did not recover in our patient and a permanent pacemaker was needed.

Further investigation into the appropriate management of arrhythmias associated with left ventricular apical ballooning syndrome and midventricular ballooning syndrome is warranted.

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